Refine Search

Search Results -

Terms	Documents
fe2+ same kidney	6

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L34

Refine Search | Recall Text | Clear | Interrupt

Search History

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Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L34	fe2+ same kidney	6	L34
L33	L29 same (kidney)	8	L33
L32	L31 same urine	4	L32
L31	fe2+	1723	L31
L30	L29 same urine	6	L30
L29	fe2$	31484	L29
L28	L27 same urine	2	L28
L27	catalytic iron	120	L27
L26	5374561.pn. and (iron) and (kidney)	1	L26
L25	(iron)near4(urine)and kidney disease	2	L25
L24	(iron)near4(urine)and kidney	30	L24
DB=EPAB; PLUR=YES; OP=ADJ			
L23	WO-200065346-A1.did.	0	L23
DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			

h e b b cg b e e ch

Refine Search

Your wildcard search against 10000 terms has yielded the results below.

Your result set for the last L# is incomplete.

The probable cause is use of unlimited truncation. Revise your search strategy to use limited truncation.

Search Results -

Terms	Documents
(measur$)near5 (iron)near15 (urin$)	16

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

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Search Results
Help
User Searches
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Logout

L4

Refine Search

Recall Text | Clear | Interrupt

Search History

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DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L4	(measur$)near5 (iron)near15 (urin$)	16	L4
DB=USPT; PLUR=YES; OP=ADJ			
L3	(measur$)near5 (iron)same (urin$)	18	L3
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L2	(iron) same (urin$) same (kidne$)	92	L2
L1	(iron) same (urine) same (kidne$)	69	L1

END OF SEARCH HISTORY

§ 3749/61 and Kidney ordering

h e b b cg b e e ch

Refine Search

Search Results -

Terms	Documents
fe near10 urine same disease	3

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L39

Refine Search

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Recall Text | Clear | Interrupt

Search History

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Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
L39	fe near10 urine same disease	3	L39
L38	fe near10 urine same kidney	1	L38
L37	fe near10 urine	60	L37
L36	fe2+ same urine	4	L36
L35	fe2+ near10 urine	1	L35
L34	fe2+ same kidney	6	L34
L33	L29 same (kidney)	8	L33
L32	L31 same urine	4	L32
L31	fe2+	1723	L31
L30	L29 same urine	6	L30
L29	fe2$	31484	L29
L28	L27 same urine	2	L28
L27	catalytic iron	120	L27
L26	5374561.pn. and (iron) and (kidney)	1	L26

h e b b cg b e e ch

L25	(iron)near4(urine)and kidney disease	2	L25
L24	(iron)near4(urine)and kidney	30	L24
DB=EPAB; PLUR=YES; OP=ADJ			
L23	WO-200065346-A1.did.	0	L23
DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L22	(iron)near4(urine)same kidney	5	L22
L21	L20 and kidney	0	L21
L20	4613616.pn.	2	L20
L19	4613616.pn. and kidney	0	L19
L18	(measur$ or analy$)near3 (iron)near7 (urine)	11	L18
DB=EPAB; PLUR=YES; OP=ADJ			
L17	RU-2166196-C1.did.	0	L17
DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L16	L15 same kidney	4	L16
L15	(iron)near5 (urine)same disease	12	L15
L14	L13 same iron	41	L14
L13	(urine)near5 (kidney)	1802	L13
L12	L9 same (kidney diseas$)	9	L12
L11	L9 same urine	6	L11
L10	l9 same kidney	35	L10
L9	hemochromatosis	469	L9
DB=USPT; PLUR=YES; OP=ADJ			
L8	hemochromatosis	313	L8
DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L7	(iron)near7 (urine)same (kidney disea$)	1	L7
DB=USPT; PLUR=YES; OP=ADJ			
L6	4684482.pn.	1	L6
DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L5	(iron level$) same (kidney)	14	L5
L4	(iron level$) near10 (kidne$)	0	L4
L3	(elevat$ or high or increas$)near3(iron)near10 (kidney)	1	L3
L2	(iron)near10 (kidney)	95	L2
L1	(iron)near10 (kidney diseas$)	2	L1

END OF SEARCH HISTORY

h e b b cg b e e ch

L22	(iron)near4(urine)same kidney	5	L22
L21	L20 and kidney	0	L21
L20	4613616.pn.	2	L20
L19	4613616.pn. and kidney	0	L19
L18	(measur$ or analy$)near3 (iron)near7 (urine)	11	L18
DB=EPAB; PLUR=YES; OP=ADJ			
L17	RU-2166196-C1.did.	0	L17
DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L16	L15 same kidney	4	L16
L15	(iron)near5 (urine)same disease	12	L15
L14	L13 same iron	41	L14
L13	(urine)near5 (kidney)	1802	L13
L12	L9 same (kidney diseas$)	9	L12
L11	L9 same urine	6	L11
L10	l9 same kidney	35	L10
L9	hemochromatosis	469	L9
DB=USPT; PLUR=YES; OP=ADJ			
L8	hemochromatosis	313	L8
DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L7	(iron)near7 (urine)same (kidney disea$)	1	L7
DB=USPT; PLUR=YES; OP=ADJ			
L6	4684482.pn.	1	L6
DB=USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L5	(iron level$) same (kidney)	14	L5
L4	(iron level$) near10 (kidne$)	0	L4
L3	(elevat$ or high or increas$)near3(iron)near10 (kidney)	1	L3
L2	(iron)near10 (kidney)	95	L2
L1	(iron)near10 (kidney diseas$)	2	L1

END OF SEARCH HISTORY

h e b b cg b e e ch

Freeform Search

Database: US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Term: (iron) same (urin$) same (kidne$)

Display: 10 **Documents in Display Format:** - **Starting with Number** 1

Generate: ○ **Hit List** ◉ **Hit Count** ○ **Side by Side** ○ **Image**

Search Clear Interrupt

Search History

DATE: Wednesday, April 28, 2004 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L2	(iron) same (urin$) same (kidne$)	92	L2
L1	(iron) same (urine) same (kidne$)	69	L1

END OF SEARCH HISTORY

First Hit Fwd Refs

Generate Collection Print

L4: Entry 2 of 16 File: USPT Sep 22, 1998

DOCUMENT-IDENTIFIER: US 5811127 A
TITLE: Desferrioxamine oral delivery system

Detailed Description Text (87):
Male Sprague-Dawley rats were anesthetized, after which their bile ducts were cannulated, such that continuous bile samples could be collected while the animals were free to move around their cages. The rats were fasted for 24 h, after which the drug was administered by gavage at a dose of 45 mg/kg. Bile samples were collected at 3-h intervals, and urine samples were taken every 24 h. The iron content of the bile and urine samples were measured by atomic absorption spectrometry essentially as described in part A.

First Hit Fwd Refs

Generate Collection Print

L4: Entry 7 of 16 File: USPT Sep 23, 1986

DOCUMENT-IDENTIFIER: US 4613616 A
TITLE: Polymeric iron chelators

Detailed Description Text (32):
On reviewing the considerable quantity of data now available on the many compounds tested, it is apparent that the most significant measure of the potency of the drug is the iron level in the urine. In the testing protocol, the percent iron change in spleen, liver, and feces are derived from small changes in materials normally fairly rich in iron. Rarely is the change more than 30% in the right direction; decrease for spleen and liver, increase for feces. Also the iron levels often vary widely with standard deviations up to 30% of the mean. On the other hand, iron levels in the urine can be increased by several hundred percent by only a moderately active iron chelator. Although standard deviations are still high, in the urine we see changes that are large and easily recognizable as significant in a material normally low in iron.

M
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U

h e b eb e h

First Hit

Generate Collection | Print

L4: Entry 9 of 16 File: JPAB Oct 22, 1976

PUB-NO: JP351120788A
DOCUMENT-IDENTIFIER: JP 51120788 A
TITLE: AUTOMATIC URINE ANALYZER

PUBN-DATE: October 22, 1976

INVENTOR-INFORMATION:

NAME	COUNTRY
NAGATA, TATSUHIKO	
FUKUMOTO, YASUHIRO	
MASAOKA, KAZUTOSHI	

ASSIGNEE-INFORMATION:

NAME	COUNTRY
TOSHIBA BETSUKUMAN KK	

APPL-NO: JP50045353
APPL-DATE: April 16, 1975

INT-CL (IPC): G01N 33/16; A61B 5/00

ABSTRACT:

PURPOSE: In an automatic urine analyzer which chemically performs measurements on hydrogen iron concentration (PH) in urine, the sample is obtained by utilizing the surface tension and by obserbing the sample with an absorption material to eliminate excessive additives for accurate measurement which has an excellent reproducibility.

h e b b g e e e f c e b e ge

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                   and searchable
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                   changes
 NEWS  6  MAR 03   MEDLINE and LMEDLINE reloaded
 NEWS  7  MAR 03   MEDLINE file segment of TOXCENTER reloaded
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                   available
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                                                      ENTRY     SESSION
FULL ESTIMATED COST                                    0.21        0.21
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The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s (iron)(P)(urin?)(P)(kidne?)
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
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PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
  26 FILES SEARCHED...
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
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FIELD CODE - 'AND' OPERATOR ASSUMED 'IRON)(P)(URIN?'
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FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
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FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
  57 FILES SEARCHED...
L1         2177 (IRON)(P)(URIN?)(P)(KIDNE?)

=> s (measur?)(5A)(iron)(10A)(urin?)(P)(kidne?)
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
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FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
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PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
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PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
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PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
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PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
  46 FILES SEARCHED...
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FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(KIDNE?'
  63 FILES SEARCHED...
L2          28 (MEASUR?)(5A)(IRON)(10A)(URIN?)(P)(KIDNE?)

=> dup rem l2
DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, ADISNEWS, BIOCOMMERCE, DGENE,
DRUGMONOG2, IMSRESEARCH, FEDRIP, FOREGE, GENBANK, IMSPRODUCT, KOSMET,
MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, RDISCLOSURE, SYNTHLINE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L2
L3           14 DUP REM L2 (14 DUPLICATES REMOVED)

=> d ibib abs l3 1-14

L3   ANSWER 1 OF 14  WPIDS  COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER:      2003-767399 [72]   WPIDS
DOC. NO. CPI:          C2003-210889
TITLE:                 Improving tissue repair of damaged tissue e.g. myocardium
                       and vascular tree comprises administering agent which
                       reduces iron values content of body .
DERWENT CLASS:         B05
INVENTOR(S):           BAKER, R J; COOPER, G J S
PATENT ASSIGNEE(S):    (PROT-N) PROTEMIX CORP LTD
COUNTRY COUNT:         103
PATENT INFORMATION:

     PATENT NO        KIND DATE     WEEK       LA   PG
     ---------------------------------------------
```

WO 2003075910 A1 20030918 (200372)* EN 53
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS
LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PH PL
PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU
ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003075910	A1	WO 2003-NZ43	20030310

PRIORITY APPLN. INFO: NZ 2002-517722 20020308
AN 2003-767399 [72] WPIDS
AB WO2003075910 A UPAB: 20031107
NOVELTY - Improving tissue repair of damaged tissue comprising the myocardium, vascular tree or organs dependent on the vascular tree comprises administering an agent which reduces the iron values content of the body.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for use of a compound which itself in vivo or which has at least one metabolite in vivo which is an iron chelator or reduces available iron values, for production of a pharmaceutical composition or dosage unit to reduce the level of iron.

ACTIVITY - Antidiabetic; Cardiant; Vasotropic.

MECHANISM OF ACTION - Iron chelator.

In a test, diabetes was induced in male Wistar rats by administering streptozocin (60 mg/kg) in citrate buffer (pH 4.5). Control animals were treated with citrate buffer and the other group (test) of animals was treated with triene (50 mg/l). Creatinine and **iron** were **measured** in **urine** samples. The **iron** (mu g/mmole creatinine) content of **urine** in test/control animals was 150/100. The results showed that triene increased urinary excretion of iron and decreased the tissue level of iron. Cardiomyopathic changes were also reduced.

USE - Used for improving repair of damaged tissue and preventing damage of tissue comprising the myocardium, vascular tree and organs dependent on the vascular tree, arising from disorders of the heart muscle (cardiomyopathy or myocarditis) (e.g. idiopathic, metabolic which includes diabetic cardiomyopathy, alcoholic, drug-induced, ischemic, and hypertensive cardiomyopathy), atheromatous disorders of the major blood vessels (e.g. aorta, coronary, carotid, cerebrovascular, renal, iliac, femoral, and popliteal arteries) (macrovascular disease), toxic, drug-induced, and metabolic (including hypertensive and/or diabetic disorders of small blood vessels (e.g. retinal arterioles, glomerular arterioles, vasa nervorum, cardiac arterioles, and associated capillary beds of the eye, **kidney**, heart, and central and peripheral nervous systems) (microvascular disease) and plaque rupture of atheromatous lesions of the major blood vessels. The method is also used for reducing elevated iron levels of the body, for treating type II diabetes mellitus, impaired glucose tolerance, heart failure, cardiomyopathy and diabetic macrovascular disease in patients not suffering from iron deficiency anemia and is not subject to classical iron overload (all claimed).

ADVANTAGE - The method decreases the chelatable iron in heart tissue and in the walls of major blood vessels avoiding iron deficiency or iron deficiency anemia. The iron chelator regimen does not generate free radicals. The improvement of the tissue repair arises from a restoration of normal tissue stem cell responses.

Dwg.0/10

L3 ANSWER 2 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 1
ACCESSION NUMBER: 2004:107736 BIOSIS
DOCUMENT NUMBER: PREV200400111413
TITLE: Altered dietary iron intake is a strong modulator of renal DMT1 expression.
AUTHOR(S): Wareing, Mark; Ferguson, Carole J.; Delannoy, Mathieu; Cox, Alan G.; McMahon, Raymond F. T.; Green, Roger; Riccardi, Daniela; Smith, Craig P. [Reprint Author]
CORPORATE SOURCE: School of Biological Sciences, Univ. of Manchester, Oxford Rd., G38, Stopford Bldg., Manchester, M13 9PT, UK craig.smith@man.ac.uk
SOURCE: American Journal of Physiology, (December 2003) Vol. 285, No. 6 Part 2, pp. F1050-F1059. print.
ISSN: 0002-9513 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Feb 2004
Last Updated on STN: 25 Feb 2004

AB Divalent metal transporter1 (DMT1; also known as DCT1 or NRAMP2) is an important component of the cellular machinery responsible for dietary iron absorption in the duodenum. DMT1 is also highly expressed in the **kidney** where it has been suggested to play a role in urinary iron handling. In this study, we determined the effect on renal DMT1 expression of feeding an iron-restricted diet (50 mg/kg) or an iron-enriched diet (5 g/kg) for 4 wk and **measured urinary** and fecal **iron** excretion rates. Feeding the low-iron diet caused a reduction in serum iron concentration and fecal iron output rate with an increase in renal DMT1 expression. Feeding an iron-enriched diet had the converse effect. Therefore, DMT1 expression in the **kidney** is sensitive to dietary iron intake, and the level of expression is inversely related to the dietary iron content. Changes in DMT1 expression occurred intracellularly in the proximal tubule and in the apical membrane and subapical region of the distal convoluted tubule. Increased DMT1 expression was accompanied by a decrease in urinary iron excretion rate and vice versa when DMT1 expression was reduced. Together, these findings suggest that modulation of renal DMT1 expression may influence renal iron excretion rate.

L3 ANSWER 3 OF 14 Elsevier BIOBASE COPYRIGHT 2004 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003289043 ESBIOBASE
TITLE: Altered dietary iron intake is a strong modulator of renal DMT1 expression
AUTHOR: Wareing M.; Ferguson C.J.; Delannoy M.; Cox A.G.; McMahon R.F.T.; Green R.; Riccardi D.; Smith C.P.
CORPORATE SOURCE: C.P. Smith, School of Biological Sciences, Stopford Bldg., Univ. of Manchester, Oxford Rd., Manchester M13 9PT, United Kingdom.
E-mail: craig.smith@man.ac.uk
SOURCE: American Journal of Physiology - Renal Physiology, (2003), 285/6 54-6 (F1050-F1059), 30 reference(s)
CODEN: AJPPFK ISSN: 0363-6127
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Divalent metal transporter1 (DMT1; also known as DCT1 or NRAMP2) is an important component of the cellular machinery responsible for dietary iron absorption in the duodenum. DMT1 is also highly expressed in the **kidney** where it has been suggested to play a role in urinary iron handling. In this study, we determined the effect on renal DMT1 expression of feeding an iron-restricted diet (50 mg/kg) or an

iron-enriched diet (5 g/kg) for 4 wk and **measured urinary** and fecal **iron** excretion rates. Feeding the low-iron diet caused a reduction in serum iron concentration and fecal iron output rate with an increase in renal DMT1 expression. Feeding an iron-enriched diet had the converse effect. Therefore, DMT1 expression in the **kidney** is sensitive to dietary iron intake, and the level of expression is inversely related to the dietary iron content. Changes in DMT1 expression occurred intracellularly in the proximal tubule and in the apical membrane and subapical region of the distal convoluted tubule. Increased DMT1 expression was accompanied by a decrease in urinary iron excretion rate and vice versa when DMT1 expression was reduced. Together, these findings suggest that modulation of renal DMT1 expression may influence renal iron excretion rate.

L3 ANSWER 4 OF 14 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2003:972319 SCISEARCH
THE GENUINE ARTICLE: 739VH
TITLE: Altered dietary iron intake is a strong modulator of renal DMT1 expression
AUTHOR: Wareing M; Ferguson C J; Delannoy M; Cox A G; McMahon R F T; Green R; Riccardi D; Smith C P (Reprint)
CORPORATE SOURCE: Univ Manchester, Sch Biol Sci, G38, Stopford Bldg, Oxford Rd, Manchester M13 9PT, Lancs, England (Reprint); Univ Manchester, Sch Biol Sci, Manchester M13 9PT, Lancs, England; Univ Sheffield, Ctr Analyt Sci, Sheffield S3 7HF, S Yorkshire, England; Univ Manchester, Fac Med, Lab Med Acad Grp, Manchester M13 9PL, Lancs, England; Manchester Royal Infirm, Dept Histopathol, Manchester M13 9WL, Lancs, England
COUNTRY OF AUTHOR: England
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-RENAL PHYSIOLOGY, (DEC 2003) Vol. 285, No. 6, pp. F1050-F1059.
Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 0363-6127.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 30
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Divalent metal transporter1 (DMT1; also known as DCT1 or NRAMP2) is an important component of the cellular machinery responsible for dietary iron absorption in the duodenum. DMT1 is also highly expressed in the **kidney** where it has been suggested to play a role in urinary iron handling. In this study, we determined the effect on renal DMT1 expression of feeding an iron-restricted diet (50 mg/kg) or an iron-enriched diet (5 g/kg) for 4 wk and **measured urinary** and fecal **iron** excretion rates. Feeding the low-iron diet caused a reduction in serum iron concentration and fecal iron output rate with an increase in renal DMT1 expression. Feeding an iron-enriched diet had the converse effect. Therefore, DMT1 expression in the **kidney** is sensitive to dietary iron intake, and the level of expression is inversely related to the dietary iron content. Changes in DMT1 expression occurred intracellularly in the proximal tubule and in the apical membrane and subapical region of the distal convoluted tubule. Increased DMT1 expression was accompanied by a decrease in urinary iron excretion rate and vice versa when DMT1 expression was reduced. Together, these findings suggest that modulation of renal DMT1 expression may influence renal iron excretion rate.

L3 ANSWER 5 OF 14 CABA COPYRIGHT 2004 CABI on STN
ACCESSION NUMBER: 2004:30951 CABA
DOCUMENT NUMBER: 20043002024
TITLE: Altered dietary iron intake is a strong modulator of renal DMT1 expression

AUTHOR: Wareing, M.; Ferguson, C. J.; Delannoy, M.; Cox, A. G.; McMahon, R. F. T.; Green, R.; Riccardi, D.; Smith, C. P.

CORPORATE SOURCE: School of Biological Sciences, G38, Stopford Bldg., University of Manchester, Oxford Rd., Manchester M13 9PT, UK. craig.smith@man.ac.uk

SOURCE: American Journal of Physiology, (2003) Vol. 285, No. 6(2), pp. F1050-F1059. 29 ref.
Publisher: American Physiological Society. Bethesda
ISSN: 0002-9513

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal

LANGUAGE: English

ENTRY DATE: Entered STN: 20040206
Last Updated on STN: 20040206

AB Divalent metal transporter1 (DMT1; also known as DCT1 or NRAMP2) is an important component of the cellular machinery responsible for dietary iron absorption in the duodenum. DMT1 is also highly expressed in the **kidney** where it has been suggested to play a role in urinary iron handling. In this study, we determined the effect on renal DMT1 expression of feeding an iron-restricted diet (50 mg/kg) or an iron-enriched diet (5 g/kg) for 4 wk and **measured urinary** and fecal **iron** excretion rates. Feeding the low-iron diet caused a reduction in serum iron concentration and fecal iron output rate with an increase in renal DMT1 expression. Feeding an iron-enriched diet had the converse effect. Therefore, DMT1 expression in the **kidney** is sensitive to dietary iron intake, and the level of expression is inversely related to the dietary iron content. Changes in DMT1 expression occurred intracellularly in the proximal tubule and in the apical membrane and subapical region of the distal convoluted tubule. Increased DMT1 expression was accompanied by a decrease in urinary iron excretion rate and vice versa when DMT1 expression was reduced. Together, these findings suggest that modulation of renal DMT1 expression may influence renal iron excretion rate.

L3 ANSWER 6 OF 14 DRUGU COPYRIGHT 2004 THOMSON DERWENT on STN

ACCESSION NUMBER: 2004-02420 DRUGU P S

TITLE: Combined early treatment with DMSA and DTPA to mobilize cadmium in rats.

AUTHOR: Blanusa M; Matek Saric M; Juresa D; Saric M; Varnai V M; Kostial K

CORPORATE SOURCE: Zagreb-Inst.Med.Res.+Occupat.Health

LOCATION: Zagreb, Croatia

SOURCE: Toxicol.Lett. (144, Suppl. 1, S135-S136, 2003)
CODEN: TOLED5 ISSN: 0378-4274

AVAIL. OF DOC.: Mineral Metabolism Unit, Institute for Medical Research and Occupational Health, Zagreb, Croatia.

LANGUAGE: English

DOCUMENT TYPE: Journal

FIELD AVAIL.: AB; LA; CT

FILE SEGMENT: Literature

AN 2004-02420 DRUGU P S

AB The influence of the chelating agents: p.o. meso-2,3-dimercapto-succinic acid (DMSA; succimer) and i.p. calcium trisodium diethylene-triaminepentaacetate (DTPA), alone and in combination, on tissue retention and distribution of cadmium after p.o. administration of cadmium chloride was compared in female albino rats. P.o. DMSA given after p.o. cadmium, removed this element very efficiently from the gastrointestinal tract. However, i.p. DTPA removes absorbed cadmium only modestly. DTPA increased zinc concentration in **kidneys** and elimination in the urine; iron and copper did not change dramatically after chelation treatment. (conference abstract: 41st Congress of the European Societies of Toxicology, EUROTOX 2003, Florence, Italy, September 28-October 1, 2003).

ABEX The dose of cadmium chloride was 0.25 mmol/kg and of chelators was 1 mmol/kg. 3 Experiments were carried out with 4 treatment groups: (1) Cd (control); (2) Cd + DMSA; (3) Cd + DTPA; and (4) Cd + DMSA + DTPA. Time intervals for chelator administration after cadmium were: immediately in the first, 30 min in the 2nd and 1 hr in the 3rd experiment. Cd, **iron**, copper and zinc were **measured** in 24-hr **urines** collected after chelator administration and in organs (liver, **kidney** and brain) at the end of each experiment. The efficiency of Cd removal from the body was lower when the time of chelator administration was longer after cadmium administration. DMSA was more efficient than DTPA. Combined chelator therapy afforded slightly better results than monotherapy. (E54/RSV)

L3 ANSWER 7 OF 14 PASCAL COPYRIGHT 2004 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2004-0124205 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2004 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Altered dietary iron intake is a strong modulator of renal DMT1 expression
AUTHOR: WAREING Mark; FERGUSON Carole J.; DELANNOY Mathieu; COX Alan G.; MCMAHON Raymond F. T.; GREEN Roger; RICCARDI Daniela; SMITH Craig P.
CORPORATE SOURCE: School of Biological Sciences, University of Manchester, Manchester M13 9PT, United Kingdom; Centre for Analytical Sciences, University of Sheffield, Sheffield S3 7HF, United Kingdom; Laboratory Medicine Academic Group, Faculty of Medicine, University of Manchester and Department of Histopathology, Manchester Royal Infirmary, Manchester M13 9WL, United Kingdom
SOURCE: American journal of physiology. Renal physiology, (2003), 54(6), F1050-F1059, 29 refs.
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-670F, 354000118779340030

AN 2004-0124205 PASCAL
CP Copyright .COPYRGT. 2004 INIST-CNRS. All rights reserved.
AB Divalent metal transporter1 (DMT1; also known as DCT1 or NRAMP2) is an important component of the cellular machinery responsible for dietary iron absorption in the duodenum. DMT1 is also highly expressed in the **kidney** where it has been suggested to play a role in urinary iron handling. In this study, we determined the effect on renal DMT1 expression of feeding an iron-restricted diet (50 mg/kg) or an iron-enriched diet (5 g/kg) for 4 wk and **measured** **urinary** and fecal **iron** excretion rates. Feeding the low-iron diet caused a reduction in serum iron concentration and fecal iron output rate with an increase in renal DMT1 expression. Feeding an iron-enriched diet had the converse effect. Therefore, DMT1 expression in the **kidney** is sensitive to dietary iron intake, and the level of expression is inversely related to the dietary iron content. Changes in DMT1 expression occurred intracellularly in the proximal tubule and in the apical membrane and subapical region of the distal convoluted tubule. Increased DMT1 expression was accompanied by a decrease in urinary iron excretion rate and vice versa when DMT1 expression was reduced. Together, these findings suggest that modulation of renal DMT1 expression may influence renal iron excretion rate.

L3 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 2000:772851 CAPLUS
DOCUMENT NUMBER: 133:307307
TITLE: Diagnosis of human **kidney** diseases by

measuring catalytic **iron** in **urine** and disease treatment with iron chelator

INVENTOR(S): Shah, Sudhir V.
PATENT ASSIGNEE(S): Shiva Biomedical, LLC, USA
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000065346	A1	20001102	WO 2000-US10775	20000421
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1173757	A1	20020123	EP 2000-928274	20000421
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002543386	T2	20021217	JP 2000-614035	20000421

PRIORITY APPLN. INFO.: US 1999-130903P P 19990423
US 1999-130908P P 19990423
WO 2000-US10775 W 20000421

AB **Kidney** disease is diagnosed by **measuring** **urinary** catalytic **iron** in humans. Progressive **kidney** disease is treated by administering an iron chelator to humans. In particular, the progression of **kidney** disease essentially can be halted and the severity of **kidney** disease can be reduced by the administration of iron chelators to humans afflicted with a progressive **kidney** disease. The methods include **measuring** catalytic **iron** content in **urine** in a human afflicted with a progressive **kidney** disease and administering an iron chelator to the human. The method can include measuring total urinary protein content, blood urea nitrogen or creatinine in a blood sample before, during or after the administration of an iron chelator. Catalytic **iron** content was **measured** in **urine** using a bleomycin assay.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 9 OF 14 NIOSHTIC on STN
ACCESSION NUMBER: 1998:4580 NIOSHTIC
DOCUMENT NUMBER: NIOSH-00240038
TITLE: Effect of Dietary Iron-Deficiency on the Disposition of Nickel in Rats
AUTHOR(S): Tallkvist, J.; Tjalve, H.
SOURCE: Toxicology Letters, Vol. 92, No. 2, pages 131-138, 18 references .
CODEN: TOLED5
PUBLICATION DATE: 21 Jul 1997
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH

AB The impact of iron deficiency on nickel (7440-02-0) disposition was investigated. Male Sprague-Dawley-rats were fed either an iron sufficient or an iron deficient diet for approximately 4 weeks. Blood samples were analyzed spectrophotometrically for hemoglobin levels. Following the diet regimen, the rats were administered a radiolabeled nickel dose of 4

micrograms per kilogram of body weight and sacrificed 3 to 120 hours later. Atomic absorption spectrometry was used to determine iron levels in various tissues. The nickel content of various tissues was determined using liquid scintillation beta-spectrometry. Over the course of 4 weeks, the hemoglobin level in iron sufficient rats increased from 9.2+/-0.7 grams per deciliter (g/dl) to 12.5+/-0.5g/dl, whereas the hemoglobin level in iron deficient rats decreased from 9.2+/-0.7g/dl to 6.2+/-0.7g/dl. Tissue iron levels were significantly higher in the iron sufficient rats than in the iron deficient rats. The body weight of iron sufficient rats was markedly higher than that of iron deficient rats. Nickel exposure through gastric intubation resulted in significantly higher tissue nickel levels in iron deficient rats than in iron sufficient rats. The relative tissue distribution of nickel was similar in iron sufficient and deficient rats, with nickel levels highest in the **kidney** and lowest in the testis. While nickel levels were also higher in iron deficient rats than in iron sufficient rats following nickel injection, the difference was not as substantial. The proportion of the injected nickel dose excreted in the **urine** 24 hours following exposure **measured** 63+/-4% in **iron** deficient rats and 79+/-5% in iron sufficient rats. The authors conclude that nickel absorption in the intestinal epithelium and nickel uptake from the blood into tissues are enhanced by iron deficiency.

L3 ANSWER 10 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 3

ACCESSION NUMBER: 1997:30650 BIOSIS
DOCUMENT NUMBER: PREV199799337053
TITLE: Effect of iron status on the intestinal absorption of aluminum: A reappraisal.
AUTHOR(S): Ittel, Thomas H. [Reprint author]; Kinzel, Silvia; Ortmanns, Annette; Sieberth, Heinz-Guenter
CORPORATE SOURCE: Dep. Internal Med., R.W.T.H., Pauwelsstrasse 30, D-52057 Aachen, Germany
SOURCE: Kidney International, (1996) Vol. 50, No. 6, pp. 1879-1888. CODEN: KDYIA5. ISSN: 0085-2538.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Jan 1997
Last Updated on STN: 28 Jan 1997

AB Clinical and experimental studies have shown that serum aluminum (Al) is bound to transferrin and that cellular uptake of Al appears to be mediated by transferrin receptors. Based on these findings it is widely believed that intestinal Al absorption occurs via iron-specific, transferrin-dependent pathways and that iron (Fe) deficiency increases the intestinal absorption of Al. However, since no transferrin receptors are expressed on the absorptive surface of small intestinal epithelial cells this notion is doubtful. To further clarify the issue the present study investigated the effect of marked alterations of body Fe stores on the intestinal absorption of Al using three different rat models. (I) Serum Al concentrations and **urinary** excretion rates of Al were **measured** in **iron**-overloaded (Fe+) or **iron** -deficient (Fe-) rats with either normal (C) or impaired (5/6 nephrectomy) renal function (Nx) employing oral Al loads in single dose studies. (11) Tissue Al accumulation as well as serum and urine Al were deter-mined in respective experimental groups exposed to a prolonged (41 days) dietary Al load. (III) To assess the effect of Fe status on the intestinal absorption of Al directly at the organ level perfusions of in situ rat gut preparations were performed. In the single dose studies administration of Al resulted in similar urinary excretion rates of Al in intact **kidney** groups (C+Fe-, 229 +- 85 nmol/5 days; C+Fe+, 240 +- 59 nmol/5 days) despite marked differences in liver Fe (C+ Fe-, 1.34 +- 0.16 vs. C+Fe+, 55.69 +- 13.20 mu-mol/g) and duodenal mucosal Fe (C+Fe-, 0.68 +- 0.11 vs. C+Fe+, 3.17 +- 0.82 mu-mol/g). In addition, mucosal Al concentration 24 hours after the load was not affected by the Fe status

(C+Fe-, 37 +- 16 nmol/g, C+Fe+, 56 +- 19 nmol/g). Regardless of the Fe status post-load Al excretion was enhanced in Nx rats (Nx+Fe-, 533 +- 234 nmol/five days, Nx+Fe+, 536 +- 201 nmol/five days). Irrespective of Fe status a prolonged dietary Al load resulted in a similar increase in tissue Al concentration (nmol/g) in liver (baseline, 159 +- 22: C+Fe-, 276 +- 125; C+Fe+, 251 +- 71; Nx+Fe-, 330 +- 119; Nx+Fe+, 437 +- 67) and in bone (baseline, 219 +- 119; C+Fe-, 433 +- 174, C+ Fe+, 485 +- 141; Nx+Fe-, 504 +- 185; Nx+Fe+, 548 +- 215). The increase in spleen Al was significantly larger in Fe-overloaded rats (baseline, 194 +- 20; C+Fe+, 511 +- 129 vs. C+Fe-, 308 +- 62, P lt 0.05; Nx+Fe+, 514 +- 67 vs. Nx+Fe-, 389 +- 119, P lt 0.05). Brain Al tended to rise in Nx rats only (baseline, 96 +- 33; Nx+Fe+, 174 +- 100, Nx+Fe-, 156 +- 78, P = NS). Analogous results were obtained in in situ intestinal perfusion studies: Fe deficiency and Fe overload both did not affect the time-dependent increase in serum Al in either systemic or portal vein blood. When paracellular intestinal permeability was assessed mannitol absorption was significantly higher in uremic animals as compared to controls. Pharmacological blockade (2 mM kinetin) of the paracellular permeability substantially reduced the time-dependent increase in serum Al in uremic rats but had little effect in control animals, suggesting that even the excess absorption of Al observed in uremia occurs via a paracellular rather than an iron-specific pathway. In conclusion, the findings of the present study provide several lines of evidence against the commonly accepted view that the intestinal absorption of Al occurs via iron-specific pathways. Most likely, this is related to the fact, that neither the absorption of Fe nor the absorption of Al are mediated via transferrin receptors. In addition, the enhanced intestinal absorption of Al observed in uremic rats does also not occur via iron-specific pathways, but seems to be due to increased paracellular permeability of the intestine.

L3 ANSWER 11 OF 14 NIOSHTIC on STN
ACCESSION NUMBER: 1997:165410 NIOSHTIC
DOCUMENT NUMBER: NIOSH-00200361
TITLE: Distribution of Terbium and Increase in Calcium Concentrations in Organs of Mice Administered with Terbium Chloride
AUTHOR(S): Shinohara, A.; Chiba, M.
SOURCE: Toxicology, Vol. 66, No. 1, pages 93-103, 24 references . CODEN: TXCYAC
PUBLICATION DATE: 11 Feb 1991
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH

AB The disposition of terbium (7440-27-9) after dosing with terbium-chloride (10042-88-3) was studied in mice. The effects of terbium on tissue calcium, magnesium, iron, and zinc concentration were also investigated. Male Crj:ICR-mice were injected intraperitoneally with 10, 50, or 250mg/kg terbium-chloride. Selected mice were killed 18 to 20 hours later to determine the tissue distribution of terbium. The tissue concentrations of calcium, magnesium, **iron**, and zinc were **measured**.
The feces and **urine** were assayed for terbium. Blood hematocrit values were determined. Other mice were maintained for 1 week and observed for mortality. Surviving mice were killed and weighed. The liver, **kidneys**, spleen, testes, lungs, and heart were removed and weighed. Approximately 80% of the mice given the 250mg/kg dose died within 1 week. The lower doses caused no deaths. Terbium-chloride caused transient dose related decreases in body weight. The 250mg/kg dose caused significant decreases in liver, **kidney**, and spleen weights. The 50 and 250mg/kg doses caused significant increases in blood hematocrit. Terbium accumulated in a dose related manner primarily in the pancreas, seminal vesicles, spleen, liver, and testes. Terbium was excreted primarily in the feces. The amounts of terbium excreted in the feces of 10 and 50mg/kg amounted to 2.19 and 1.08% of the doses, respectively. Sufficient feces for analysis was not excreted by mice given 250mg/kg

terbium. Terbium cause dose dependent increases in calcium concentration in the liver, spleen, pancreas, seminal vesicles, and testes. The terbium and calcium concentrations in these organs were well correlated, the best correlation occurring in the liver. Terbium induced small increases in magnesium concentration in most tissues. The only effect on tissue iron was in the spleen where the 250mg/kg dose caused a significant decrease. The 50 and 250mg/kg doses caused significant increases in hepatic zinc content. Zinc concentrations in the other organs were not affected by terbium. The authors conclude that terbium accumulation and induction of calcium in body tissues appear to be related.

L3 ANSWER 12 OF 14 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 4

ACCESSION NUMBER: 86105563 EMBASE
DOCUMENT NUMBER: 1986105563
TITLE: Essential trace metal excretion from rats with lead exposure and during chelation therapy.
AUTHOR: Victery W.; Miller C.R.; Goyer R.A.
CORPORATE SOURCE: National Institute of Environmental Health Sciences, Research Triangle Park, NC 27709, United States
SOURCE: Journal of Laboratory and Clinical Medicine, (1986) 107/2 (129-135).
CODEN: JLCMAK
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
052 Toxicology
029 Clinical Biochemistry
030 Pharmacology
LANGUAGE: English

AB **Urinary** excretion of lead, zinc, calcium, magnesium, **iron**, copper, sodium, and potassium was **measured** in rats daily for 1 week after a 6-week exposure to 10,000 μg/ml lead in drinking water. Beginning on the third day, half of the lead-exposed and control rats were injected intraperitoneally with calcium disodium ethylenediaminetetraacetate (EDTA) daily for 3 days. Whole blood, plasma, and **kidney** metal concentrations were determined from samples obtained at the end of the experiment. Exposure to lead increased urinary excretion, not only of lead, but also of calcium, magnesium, zinc, copper, and iron. Excretion of sodium and potassium was not altered. Chelation therapy further increased excretion of lead, zinc, copper, and iron, but not magnesium. The increase in calcium excretion during chelation treatment (beyond that resulting from lead exposure per se) was accounted for by the Ca content of CaNa2-EDTA. EDTA treatment increased renal concentration of zinc but lowered renal concentration of lead, copper, and iron. These multimetal alterations may have implications for essential metal supplementation, particularly zinc, in persons being given chelation agents for excess lead exposure and in infants and children with low-level lead exposure not necessarily requiring chelation therapy.

L3 ANSWER 13 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 5

ACCESSION NUMBER: 1986:206224 BIOSIS
DOCUMENT NUMBER: PREV198681097524; BA81:97524
TITLE: ESSENTIAL TRACE METAL EXCRETION FROM RATS WITH LEAD EXPOSURE AND DURING CHELATION THERAPY.
AUTHOR(S): VICTERY W [Reprint author]; MILLER C R; GOYER R A
CORPORATE SOURCE: NATL INST ENVIRON HEALTH SCI, PO BOX 12233, RESEARCH TRIANGLE PARK, NC 27709, USA
SOURCE: Journal of Laboratory and Clinical Medicine, (1985) Vol. 107, No. 2, pp. 129-135.
CODEN: JLCMAK. ISSN: 0022-2143.
DOCUMENT TYPE: Article
FILE SEGMENT: BA

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L5: Entry 8 of 14 File: USPT Sep 10, 1991

DOCUMENT-IDENTIFIER: US 5047421 A
TITLE: Orally effective ion chelators

Brief Summary Text (24):
Compounds of formula I are prodrug forms of deferoxamine which liberate deferoxamine in the body to complex and/or chelate iron for subsequent excretion when administered to a human being, and are therefore useful in therapy in the treatment of diseases in which iron levels in the body have elevated or toxic levels. These diseases include, for example, thalassemia major, sideroachrestic anemia, Blackfan-Diamond anemia, aplastic anemia, sickle cell anemia, hemolytic anemias and hemosiderosis brought about by multiple blood transfusions or such condition when brought about by treatment of an anemia found in kidney-damaged patients undergoing renal dialysis.

LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 28 May 1986
Last Updated on STN: 28 May 1986

AB **Urinary** excretion of lead, zinc, calcium, magnesium, **iron**, copper, sodium, and potassium was **measured** in rats daily for 1 week after a 6-week exposure to 10,000 μg/ml lead in drinking water. Beginning on the third day, half of the lead-exposed and control rats were injected intraperitoneally with calcium disodium ethylenediaminetetraacetate (EDTA) daily for 3 days. Whole blood, plasma, and **kidney** metal concentrations were determined from samples obtained at the end of the experiment. Exposure to lead increased urinary excretion, not only of lead, but also of calcium, magnesium, zinc, copper, and iron. Excretion of sodium and potassium was not altered. Chelation therapy further increased excretion of lead, zinc, copper, and iron, but not magnesium. The increase in calcium excretion during chelation treatment (beyond that resulting from lead exposure per se) was accounted for by the Ca content of CaNa2-EDTA. EDTA treatment increased renal concentration of zinc but lowered renal concentration of lead, copper, and iron. These multimetal alterations may have implications for essential metal supplementation, particularly zinc, in persons being given chelation agents for excess lead exposure and in infants and children with low-level lead exposure not necessarily requiring chelation therapy.

L3 ANSWER 14 OF 14 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1984:15183649 BIOTECHNO
TITLE: Persistence of inert macromolecules (imposil) in the rat mesangium and glomerular functional disturbance
AUTHOR: Goode N.P.; Davison A.M.; Gowland G.; et al.
CORPORATE SOURCE: Renal Research Unit, St. Jame's University Hospital, Leeds, United Kingdom.
SOURCE: Journal of Pathology, (1984), 144/3 (179-187)
CODEN: JPTLAS
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English

AN 1984:15183649 BIOTECHNO

AB Imposil iron-dextran is an inert tracer that has been used to study mesangial uptake and clearance of macromolecular material from the glomerular circulation. Such a tracer may be a useful marker of altered mesangial function in animals with some forms of glomerulonephritis. We have studied mesangial handling of intravenously injected Imposil (50 mg/100 g body weight) in normal rats by light, immunofluorescence and electron microscopy for up to 3 months. Mesangial cell uptake was maximal at 45-54 h. Extrusion and drainage of tracer to the vascular pole and distal tubule was evident at 3 days but iron was still present in mesangial cells at 3 months. Possible functional renal impairment resulting from persistent mesangially sequestered tracer was examined by **measuring** daily **urine** protein and **iron** excretion. A possible relationship between failure of mesangial cells to eliminate inert tracer and increasing glomerular permeability is demonstrated, suggesting that Imposil and similar inert macromolecules cannot be used for long-term studies of mesangial function.

L2: Entry 22 of 92 File: USPT Jan 29, 2002

DOCUMENT-IDENTIFIER: US RE37534 E
TITLE: Pharmaceutical compositions

Detailed Description Text (32):
Identical experiments carried out with .sup.59 Fe labelled iron(III) EDTA gave a entirely different picture as will be seen for the results of a typical experiment illustrated in FIG. 6 (in which the lower end of the ordinate represents the background level) and Table 6 (the amount of .sup.59 Fe administered in this experiment was 2 .mu.Ci but the figures given in the table have been adjusted to correspond to a dosage of 2.2.times.10.sup.6 cpm in order to facilitate comparison with Table 5). In this experiment the radioactivity in the blood showed no initial plateau. Instead, loss of radioactivity followed at least a two-component process such that a large amount found its way to the urine rather than to the issues. The rate constant of the elimination from the blood to the linear phase of the regression was 0.023/minute. The concentration of radioactivity in the kidney and urine, and not in the bone marrow or spleen, would indicate that iron in this form does not appear to be able to attach to transferrin in the plasma and protect itself from urinary excretion. The combined tissue of heart, liver and spleen contained only 1% of the original dose at the end of the experiment, whereas the urine contained over 50%. This is in accord with the fact that EDTA does not exchange iron with transferrin rapidly.

Detailed Description Text (33):
The iron maltol complex (100 .mu.g Fe) was also administered to the duodenum of the cat in the presence of a 40 fold excess of maltol followed by 5 ml of 150 ml tris hydrochloride buffer (pH 7.4). In this case the .sup.59 Fe content of the blood, as shown in FIG. 7, reaches a maximum level 2 hours after the initial administration (the readings start at about 300 cpm/0.5 ml which represents the background reading). The distribution of .sup.59 Fe in the tissues of the animal after the same duodenal experiment to which FIG. 7 relates were investigated and the typical results are shown in Table 7. The amount of 59Fe and administered in this experiment was 10 .mu.Ci or 5.327.times.10.sup.6 cpm into a 2.9 kg cat. It will be seen that the distribution of the .sup.59 Fe after 4 hours was similar to that after intravenous infusion, with low levels in the kidney and urine and high levels in both the spleen and bone marrow.

Detailed Description Paragraph Table (5):
TABLE 5 (Iron maltol, i.v.) Sample Net .sup.59 Fe Net total .sup.59 Fe Total tissue weight content content Tissue weight (g) (g) (cpm/g) (cpm) Heart 14.4 0.91 490 7,056 Liver 105 1.3 510 53,550 Spleen 8.4 0.86 14,890 125,076 Kidney 12.2 1.05 546 6,661 Skeletal muscle -- 1.85 0 0 Sternum -- 1.2 3,200 -- (bone marrow) Urine -- 1 152 <3,000

Detailed Description Paragraph Table (6):
TABLE 6 (Iron EDTA, i.v.) Sample Net .sup.59 Fe Net total .sup.59 Fe Total tissue weight content content Tissue weight (g) (g) (cpm/g) (cpm) Heart 15.5 1.01 209 3,248 Liver 75 1.21 261 19,600 Sternum -- 0.28 1,164 -- (bone marrow) Spleen 11.1 0.89 162 1,814 Kidney 19.2 1.47 1,134 21,770 Skeletal muscle -- 2.59 95 -- Urine 19 ml 2 ml 62,156 1,180,900

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Detailed Description Paragraph Table (7):
TABLE 7 (Iron maltol, per duodenum) Sample Net .sup.59 Fe Net total .sup.59 Fe Total tissue weight content content Tissue weight (g) (g) (cpm/g) (cpm) Heart 14 0.633 50 1,106 Liver 81 1.45 400 32,400 Spleen 12.7 1.19 3,783 48,047 Kidney 14.4 0.835 79 1,138 Sternum 10 1.26 790 7,905 (bone marrow) Bile .about.5 ml 1 ml 2,200 .about.11,000 Urine .about.10 ml 1 ml 22 .about.220

Detailed Description Paragraph Table (8):
TABLE 8 (Iron EDTA, per duodenum) Sample Net .sup.59 Fe Net total .sup.59 Fe Total tissue weight content content Tissue weight (g) (g) (cpm/g) (cpm) Heart 15.3 1.18 188 2,878 Liver 59.3 0.78 499 29,574 Kidney 11.3 0.90 1,762 19,913 Spleen 4.4 0.42 200 880 Sternum -- 0.78 917 -- (bone marrow) Skeletal muscle -- 1.48 117 -- Urine 15 ml 5 ml 36,306 544,596

L2: Entry 52 of 92 File: USPT Jul 2, 1991

DOCUMENT-IDENTIFIER: US 5028411 A
** See image for Certificate of Correction **
TITLE: Pharmaceutical compositions

Detailed Description Text (32):
Identical experiments carried out with .sup.59 Fe labelled iron(III) EDTA gave a entirely different picture as will be seen for the results of a typical experiment illustrated in FIG. 6 (in which the lower end of the ordinate represents the background level) and Table 6 (the amount of .sup.59 Fe administered in this experiment was 2 .mu.Ci but the figures given in the table have been adjusted to correspond to a dosage of 2.2.times.10.sup.6 cpm in order to facilitate comparison with Table 5). In this experiment the radioactivity in the blood showed no initial plateau. Instead, loss of radioactivity followed at least a two-component process such that a large amount found its way to the urine rather than to the tissues. The rate constant of the elimination from the blood of the linear phase of the regression was 0.023/minute. The concentration of radioactivity in the kidney and urine, and not in the bone marrow or spleen, would indicate that iron in this form does not appear to be able to attach to transferrin in the plasma and protect itself from urinary excretion. The combined tissue of heart, liver and spleen contained only 1% of the original dose at the end of the experiment, whereas the urine contained over 50%. This is in accord with the fact that EDTA does not exchange iron with transferrin rapidly.

Detailed Description Text (33):
The iron maltol complex (100 .mu.g Fe) was also administered to the duodenum of the cat in the presence of a 40 fold excess of maltol followed by 5 ml of 150 ml Tris hydrochloride buffer (pH 7.4). In this case the .sup.59 Fe content of the blood, as shown in FIG. 7, reaches a maximum level 2 hours after the initial administration (the readings start at about 300 cpm/0.5 ml which represents the background reading). The distribution of .sup.59 Fe in the tissues of the animal after the same duodenal experiment to which FIG. 7 relates were investigated and the typical results are shown in Table 7. The amount of .sup.59 Fe administered in this experiment was 10 .mu.Ci or 5.327.times.10.sup.6 cpm into a 2.9 kg cat. It will be seen that the distribution of the .sup.59 Fe after 4 hours was similar to that after intravenous infusion, with low levels in the kidney and urine and high levels in both the spleen and bone marrow.

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6,661 Skeletal muscle -- 1.85 0 0 Sternum -- 1.2 3,200 -- (bone marrow) Urine -- 1
152 <3,000

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L2: Entry 61 of 92 File: USPT Oct 29, 1985

DOCUMENT-IDENTIFIER: US 4550101 A

TITLE: Iron complexes of hydroxy pyridones useful for treating iron deficiency anemia

Detailed Description Text (41):

The action of the iron complex of 1-ethyl-3-hydroxypyrid-2-one, prepared as described in Example 1, was compared with that of iron (III) EDTA (1:1 molar ratio) which is one of the iron compounds currently marketed for the treatment of iron deficiency anaemia. Two cats (ca 2.7 kg) were anaethetised and a solution in aqueous tris hydrochloride (20 mM, pH 7.4) of .sup.59 Fe labelled complex (1 ml of solution with an iron concentration of 1 mg/1 ml) was administered directly into the small intestine, one cat receiving iron (III) 1-ethyl-3-hydroxypyrid-2-one, in the presence of a 20-fold excess of metal-free 1-ethyl-3-hydroxypyrid-2-one, and the other receiving iron (III) EDTA. The blood levels of .sup.59 Fe were recorded as a function of time, the results being shown in Table 4, and both cats were then killed after 4 hours. The total .sup.59 Fe present in the kidneys and urine was then measured in each case. The percentage of the total dose in the kidneys and urine was 1.6% and 21%, respectively, for the iron (III) EDTA indicating that this complex is cleared rapidly into the urine. For the iron (III) 1-ethyl-3-hydroxypyrid-2-one, however, 0.05% of the total dose was found in the kidneys and no more than 0.01% in the urine, clearance in this manner therefore being very slow for this complex.

Detailed Description Text (42):

As well as the kidneys, various other tissues of the cat which recieved the Fe(III) 1-ethyl-3-hydroxypyrid-2-one were studied to assess the percentage of the original .sup.59 Fe dose which was present. The results are shown in Table 5 and it will be seen that the iron, rather than being excrete in the urine, is instead widely distributed throughout the body. Furthermore, when this neutral complex was injected intravenously it was cleared from the circulation with a half life of 45 minutes. The tissue distribution after intravenous injection was found to be similar to that resulting from the intrajejunal infusion, these results also being reported in Table 5, from which it will be seen that in this instance less than 1% of the dose appears in the urine. The bulk of the dose given by either route is estimated to be in the reticuloendothelial system (bone marrow). This was confirmed by the identification of high levels of .sup.59 Fe in the sternum. Over 95% of the .sup.59 Fe present in the blood after 1 hour was bound to transferrin and thus would be expected to be mainly directed to the reticuloendothelial system.

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L2: Entry 68 of 92 File: EPAB Mar 12, 1998

DOCUMENT-IDENTIFIER: WO 9809626 A1
TITLE: METHODS FOR IN VIVO REDUCTION OF IRON LEVELS AND COMPOSITIONS USEFUL THEREFOR

Abstract Text (1):
In accordance with the present invention, there are provided methods for the in vivo reduction of free iron ion levels in a mammalian subject. The present invention employs a scavenging approach whereby free iron ions are bound in vivo to a suitable physiologically compatible scavenger. The resulting complex renders the free iron ions harmless, and is eventually excreted in the urine of the host. Further in accordance with the present invention, there are provided compositions and formulations useful for carrying out the above-described methods. An exemplary scavenger contemplated for use in the practice of the present invention is a dithiocarbamate-containing composition. This material binds to free iron ions, forming a stable, water-soluble dithiocarbamate-iron complex. Thus, the present invention relates to methods for reducing in vivo levels of free iron ions as a means of treating subjects afflicted with iron overload and non-iron overload diseases and/or conditions, such as thalassemia, anemia, hereditary hemochromatosis, hemodialysis, stroke and rheumatoid arthritis. Dithiocarbamate-containing scavengers are administered to a host in need of such treatment; these scavengers interact in vivo with free iron ions, thereby forming a stable dithiocarbamate-metal complex, which is then filtered through the kidneys, concentrated in the urine, and eventually excreted by the subject, thus reducing in vivo levels of free iron ions.

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